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Gallbladder Agenesis in a Miniature Pinscher : Computed Tomographic and Ultrasonographic Features

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Abstract : A 4-year-old spayed female Miniature Pinscher was presented with a 3.5-year history of elevated liver enzymes level. Continuous elevation of liver enzymes was found on repeated blood examinations and the dog was referred for further evaluation. The abdominal ultrasonography revealed that the gallbladder could not be visualized in any plane. The absence of gallbladder was suspected on ultrasonography. Computed tomography (CT) confirmed gallbladder agenesis (GBA). This is the first reported case of diagnosis of GBA in Miniature Pinscher by using CT.

Key words **:** Computed tomography, Dog, Gallbladder agenesis, Miniature Pinscher.

Introduction

Based on the previous reports (2,15), prevalence of gallbladder agenesis (GBA) is an extremely rare both in humans and dogs. GBA in humans is generally found incidentally during either abdominal surgery or autopsy. Until recently GBA in dogs has been reported in only two young female Maltese dogs (2,15) and in a Chihuahua (13). Two of the reported dogs with GBA were relatively young (2,15) and one dog were 4-year-old when diagnosed (13). The cases of previous reports are diagnosed by exploratory laparotomy (2,13,15) and retrograde cholangiography (13). The etiology of GBA is unknown both in dogs and humans, but in humans it is generally thought to be a developmental failure of the pars cystic from which the gallbladder arises during embryogenesis (13).

We report a case of a Miniature Pinscher with long-term elevation of alanine transferase (ALT) levels, because of GBA, which was confirmed by computed tomography (CT) after being on the basis of ultrasonographic findings.

Case

A 4-year-old spayed female Miniature Pinscher weighing 3.44 kg was examined by the referring veterinarian for a health screening. Blood examination revealed elevated ALT (507 U/L; reference range, 17-78 U/L) and alkaline phosphatase (ALP: 357 U/L; reference range, 72-265 U/L) levels. However, the findings of complete blood count were unremarkable. Ursodeoxycholic acid (10 mg/kg, PO, q 24 hrs, Korea United Pharm, Seoul, Korea), *S*-adenosylmethionine (7.5 mg/kg, PO, q 24 hrs, YooYoung Pharm, Seoul, Korea), and glutathione (1-2 g/dog PO q 24 hrs, Dong-A Pharm, Seoul, Korea) were initially prescribed. At the follow-up evaluations, the levels of liver enzymes continued to increase (until at 41 months after the first admission: ALT > 1,000 U/L; ALP, 585 U/L; aspartate aminotransferase (AST), 77 U/L; reference range, 15-43 U/L) even though the dog was clinically normal. The dog was then referred to the Veterinary Medical Teaching Hospital of Konkuk University. On presentation, the dog was alert, and physical examinations revealed no abnormalities. The findings of complete blood count were within normal limits. Serum biochemistry profiles revealed marked elevation in the levels of liver enzymes: ALT, 956 U/L (reference range, 19-70 U/L); AST, 140 U/L (reference range, 15-43 U/L); ALP, 208 U/L (reference range, 15-127 U/L); and gamma-glutamyltranspeptidases (GGT), 44.3 U/L (reference range, 0-6.0 U/L). Fasting ammonia levels were normal (18.0 umol/L; reference range, 0-92 umol/L). Activated partial thromboplastin time, prothrombin time, and antithrombin III and D-dimer levels were within normal limits. Fasting bile acid levels were elevated (131.5 μmol/L; reference range, 0-10.0 μmol/L), but the postprandial bile acid levels were within normal limits (16.2 μmol/L; reference range, 0-25.0 μmol/L). The polymerase chain reaction (PCR) test results were negative for virus infection (canine parvovirus, canine distemper virus, canine adenovirus, canine corona virus, canine herpes virus). Thoracic and abdominal radiographs showed mild microhepatica. Abdominal ultrasonography revealed normal morphology and echogenicity of the liver. But the gallbladder could not be visualized in any plane (Fig 1B); other abdominal organs appeared normal. The normal gallbladder is seen as a round or oval echo free structure on transverse scan (Fig 1A) and measures less than 3.5 mm in its widest

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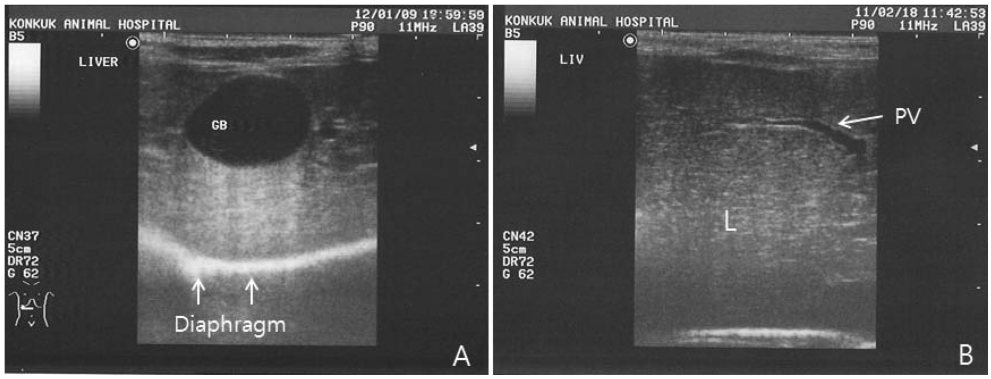

Fig 1. Ultrasonography of the abdomen showing the liver. A: Transverse plane of the gallbladder in normal dog. The gallbladder is seen on the midline. B: By using a transverse plane in the case, the liver (L) is entirely scanned, the gallbladder is absence. Branching portal veins (PV) with hyperechoic boundaries are also recognized.

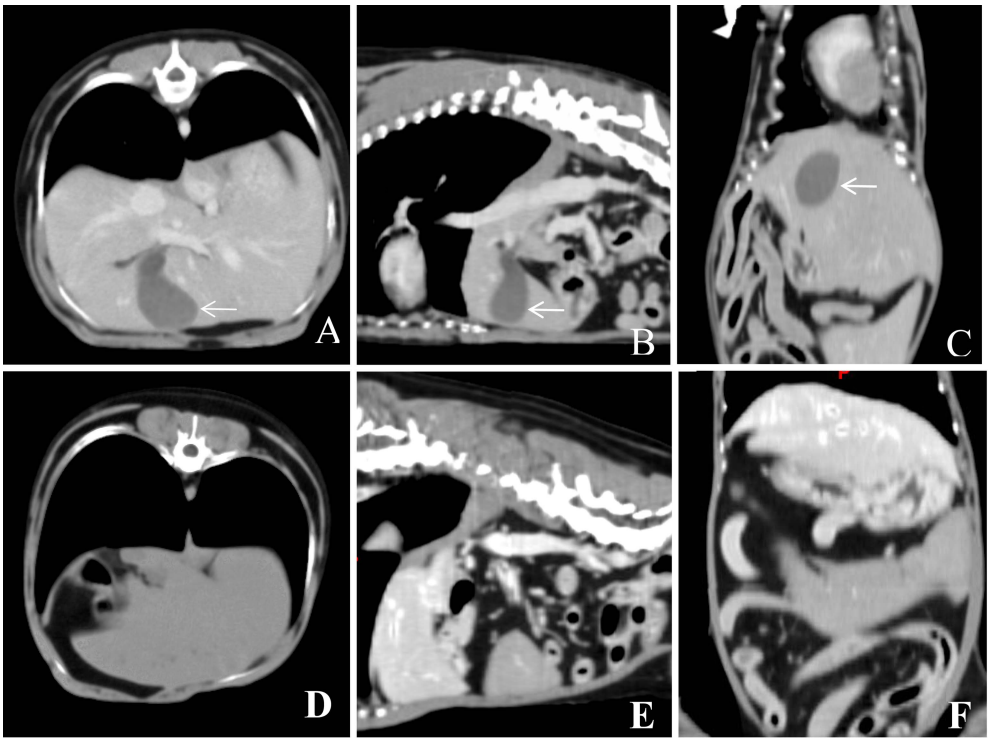

Fig 2. Computed tomography scan of the abdomen showing the gallbladder (white arrow). Normal gallbladder position of a normal dog. (A) Transverse image. (B) Sagittal image. (C) Dorsal image. Computed tomography scan of the abdomen showing absence of the gallbladder in this case. There were no cystic lesions in the omentum; falciform ligament; or intrahepatic, retroperitoneal, retrohepatic, or retroduodenal region. (D) Transverse image. (E) Sagittal image. (F) Dorsal image.

diameter. On incidental finding, calculi were observed in the urinary bladder (size, 9.6 mm × 2.3 mm and 7.1 mm × 2.1 mm) and the left kidney (size, 4.1 mm × 2.0 mm). Abdominal CT (Asteion 4®, Toshiba, Japan) scan was performed to further identify anatomical abnormalities. Fine needle aspiration of liver parenchyma revealed no remarkable findings. CT scan of the abdomen of the Miniature Pinscher obtained after injecting an intravenous non-ionic contrast medium (Omnipaque; GE Healthcare, Milwaukee, WI, USA, 280 mgI/ml) did not show the gallbladder (Fig 2D-2F); When compared with CT image of a normal dog (Fig 2A-2C), no cystic lesions were found in the lesser omentum; falciform ligament; or intrahepatic, retroperitoneal, retrohepatic, or retroduodenal region (Fig 2D-2F). A diagnosis of gallbladder agenesis was confirmed based on lack of a gallbladder on ultrasonography and on CT scan. The dog was prescribed with a low-fat diet and discharged from the hospital. The dog was given *S*-adenosylmethionine (7.5 mg/kg, PO, q 24 hrs) on trial basis; however, ALT (994 U/L; reference range, 12-118 U/L), GGT (16 U/L; reference range, 1-12 U/L), and AST

(85 U/L; reference range, 15-66 U/L) concentrations remained to be elevated. The *S*-adenosylmethionine treatment was discontinued, and the dog has been asymptomatic for 13 months after referral. The owners declined additional diagnostic tests on the dog.

Discussion

GBA is an extremely rare abnormality in both humans and dogs (5). Three types of GBA have been described in humans. The first type is GBA associated with other congenital abnormalities, which can be identified early in life (5,9). The second type is asymptomatic GBA. This type is found incidentally on autopsy in most cases. 77% of GBA patients are estimated to have asymptomatic GBA (5). The third type is GBA characterised by pain in the right upper abdominal region, nausea, and intolerance to fatty foods. This type of GBA has mostly been reported in adults (5,8,11). The exact etiology of GBA is unknown, but it is generally considered a congenital abnormality. In symptomatic humans with GBA, the most common clinical signs are pain in the right upper quadrant of the abdomen, nausea, vomiting, fatty-food intolerance, dyspepsia, and jaundice (3,7). These clinical signs could be caused by biliary dyskinesia or could develop secondary to ascending cholangitis (1,4,11). In the previous three reports of canine GBA, the clinical signs are asymptomatic (2), anorexia and vomiting (15), intermittent vomiting of bile (13). In our case, the dog showed asymptomatic despite elevated liver enzymes. For relieving elevated hepatic enzymes we used ursodeoxycholic acid to prevent progression of further biliary stasis and glutathione to protect hepatocytes from oxidative stress. In addition, S-adenosylmethionine was used for the purpose of hepatoprotection. The previous report in veterinary literature reported marked elevation of ALT and variable increase of AST, ALP, and GGT (2,13,15). In the case presented here, similar results of serum biochemical panels were observed. The most likely causes of elevated ALT in young dogs, without clinical signs include infectious/inflammatory liver diseases, hepatic anoxia, trauma, portosystemic shunt, biliary obstruction, and exposure to toxins (14). In our case, the significance and mechanisms of elevated liver enzymes have not been completely understood. However, it can suggest that the absence of gallbladder leads to failure of intermittent excretion of bile into the duodenum. This failure of bile excretion may result in biliary dyskinesia and, with dysfunctional Oddi sphincter, reflux of duodenal contents (5,6,11), leading to cholangiohepatitis. The consequent biliary inflammation and hepatitis may increase ALT and AST levels (5). In our case, histological examination was not performed. But fine needle aspiration of liver parenchyma revealed no remarkable findings.

In the present case, fasting total serum bile acid (TSBA) concentrations (131.5 μmol/L; reference range, 0-10.0 μmol/L) were higher than postprandial value (16.2 μmol/L; reference range, 0-25.0 μmol/L). A previous report (13) showed similar results of bile acid test with this case. This happens when interdigestive gallbladder contraction occurs during the course of the fast preceding the test. It may also be associated with individual variations in gastric emptying, response to cholecytokinin release, and intestinal transmit time (14). The absence of a gallbladder results in the continuous excretion of bile into the duodenum. The concentration of TBSA could be therefore high due to the lack of storage and absorption of bile in the gallbladder.

Clinical markers for GBA are yet to be defined; in the cases of previous reports, GBA was suspected at ultrasonography and confirmed by exploratory laparotomy (2,15) and retrograde cholangiography (13). In the present case, exploratory laparotomy and retrograde cholangiography was not performed, but we conducted computed tomography for confirming gallbladder agenesis. A possible diagnosis of GBA in human with non-visualization GB on US may be confirmed by cholangiography, abdominal CT scan, laparoscopy for exploration (8), endoscopic retrograde cholangiopancreatography (ERCP), or magnetic resonance cholangiography (MRC). These should be considered adequate modalities for the diagnosis of GBA, without the need for the formal laparotomy or laparoscopy with through exploration (1,7,12). This will allow avoidance of invasive, inspecific instances, by judicious use of these newer investigative modalities for confirmation of diagnosis. In humans, among nine patients with GBA, only abdominal CT scan was able to accurately determine the condition preoperatively (7). If the GB is not visualized on US, the possibility of gallbladder agenesis must be considered. However, possible ectopic locations of the gallbladder may be present in an abnormal location. Ectopic gallbladders can be located intrahepatically, on the left side between the leaves of the lesser omentum, retroperitoneally or retrohepatically, within the falciform ligament, or in the retroduodenal or retropancreatic area (10). In our case, no cystic lesion was noted in these sites visualized by CT. We can exclude the possibility of ectopic gallbladder in our patient, based on the CT findings although we did not perform endoscopic retrograde cholangiography (ERC), laparoscopy, laparotomy for exploration. The possibility of gallbladder atrophy developed secondary to cholecystolithiasis in our patient could also be ruled out because there was no cholecystolithiasis observed in this case.

In conclusion, this cased demonstrates that GBA in a dog can be more easily visualized than previous conventional diagnostic tools if we use abdominal CT scan.

Acknowledgements

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담낭 무형성을 가진 미니어쳐 핀셔에서 컴퓨터단층촬영과 초음파 특징

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요 약 : 4살령의 중성화된 암컷 미니어쳐 핀셔가 3년 5개월 동안의 지속적인 간효소 수치 상승으로 본원으로 내원하였다. 간효소 수치의 지속적인 상승은 반복적인 혈액검사를 통해 확인하였으며, 간효소 수치 상승 원인에 대한 정확한 진단을 위하여 추가적인 검사가 진행 되었다. 복부초음파검사에서 복강내의 어느 단면에서도 담낭을 확인할 수 없었다. 복부초음파검사에서 담낭의 무형성을 의심하였으며 컴퓨터단층촬영을 통해 담낭 무형성을 확인하였다. 이 증례는 미니어쳐 핀셔에서 컴퓨터단층촬영을 이용하여 담낭 무형성을 진단한 첫번째 증례보고이다.

주요어 : 컴퓨터단층촬영, 개, 담낭 무형성, 미니어쳐 핀셔